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If you are having trouble reading or viewing the ebook, please [click here](#) for help. Drug-induced liver injury (DILI) is one of the most frequent adverse drug reactions, and approximately 3%-7% of all patients will experience serious DILI during their lifetime. DILI has long been considered idiosyncratic, meaning individual in nature and unanticipated. However, the application of the generic linear no-threshold model suggests that potentially low-dose exposures lead to reactive metabolites, which, on their own, can result in liver injury. Multiple animal and human studies demonstrate that 10-50% of the population will develop idiosyncratic DILI. Therefore, it is possible that a small percentage of patients with DILI will be highly susceptible to the drug because of genetic polymorphisms affecting drug metabolism or toxicity. Identifying these patients with potential risk is a challenge that is compounded by a paucity of predictive biomarkers of DILI susceptibility. Here, we propose that a model of idiosyncratic DILI is uni-directional. While low-dose exposures produce reactive metabolites, which on their own can cause liver injury, immunosuppression underlies the induction of high-level hypersensitivity. These low-dose exposures can only produce hypersensitivity if the patient is able to mount an inflammatory response, which itself requires the presence of functional Toll-like receptors (TLRs). This is a somewhat counterintuitive hypothesis; however, we have evidence that while mice are considered hyporesponsive to a low immunogenic stimulus, treatment with low-dose nonsteroidal anti-inflammatory drugs will lead to activation of their innate immune system. We have performed a screen in mice for genetic variants of relevance to immunosuppression-driven hypersensitivity DILI. We discovered that mice with mutations in cytokines associated with immunosuppression had an increased risk of DILI. We are currently developing more in-depth validation studies and are optimistic that this model of immunosuppression-driven hypersensitivity can be exploited to uncover the genetic polymorphisms predisposing individuals to idiosyncratic DILI. Novel information and insights from these studies will provide critical insights into the susceptibility of individual patients to idiosyncratic DILI. From a clinical perspective, this has implications for the use of the generic LNT model in DILI risk prediction. Pharmacokinetic differences will alter the amount of reactive metabolites in a clinically relevant concentration. A recent study demonstrated that a pharmacokinetic difference of 1  $\mu\text{g/mL}$  is associated

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